# ACUTE ANTERIOR POLIOMYELITIS IN A YOUTH.

By Theodore A. Hoch, M.D.,

ASSISTANT PHYSICIAN AND PATHOLOGIST, WORCESTER INSANE HOSPITAL,
WORCESTER, MASS.

(*Continued from page* 557.)

The anterior root zone shows numerous blackened areas by the Marchi method, a scarcity of nerve fibers and a beginning sclerosis. This degeneration is less marked in the lateral columns. In the external part of the posterior columns there is a diffuse, though well-marked area of degeneration which is rather more sharply defined in the dorsal region. The white matter below the fourth lumbar segment does not appear unusual though the gray matter still contains small areas of destruction.

*Etiology.*—There is a very wide range in the etiology of this disease, varying from slight colds to the most severe infectious diseases. Age, season, trauma and infectious diseases are the most important etiological factors. The average age of onset is 3 years. Most cases occur under 10, and four-fifths of these under 3. It may, however, begin as late as 60, but most adult cases appear before 30. It will be seen that by far the greatest number of cases develop at the age when the child learns to walk; that is, when the spinal centres are unusually active and consequently more susceptible to toxic and other accidental causes which may be present. It is in the hot summer months when the disease finds most of its victims, more especially from June to September. Most of the large epidemics have occurred during these months.

*Trauma.*—Traumatic influences are perhaps more potent than a hasty review of the literature would lead us to believe. Several cases have been reported which followed falls and injuries directly. Bullen[7] reported a case of paralysis of both legs. Two years later a weight fell upon the patient's left hand which soon became paralyzed.

Two cases of traumatic origin are described by von Leyden[28]; one in a man, æt. 63, whose leg was fractured at the age of 2, followed by arrested development of the leg. A sclerotic lesion was found in the gray matter of the cervical cord. In the second case, paralysis of the leg followed an injury to the foot.

Stark[53] mentions the case of a woman who fell from a wagon, striking on her back and right shoulder. The injury was soon followed by weakness of the right shoulder and arm which terminated in paralysis. The anatomical changes in this case were characteristic of poliomyelitis with pronounced parenchymatous changes, but only slight evidence of inflammation. A case of injury to the arms, followed 6 months later by progressive weakness of the right hand and arm and a diffuse amyotrophy with reaction of degeneration is cited by Perrin.[40] One year later the left arm became involved, and finally the trunk and legs.

Burckhardt[8] reports a case of a boy, 1 year old, whose face was scalded with boiling water. He developed vomiting and fever in a few days, with paralysis of all the extremities and trunk muscles. He died four days after the accident, and the cord showed macroscopical and microscopical changes in the gray matter.

E. Meyer[31] observed a progressing paralysis of the right leg following injury to the right foot in a man 59 years of age. Paralysis of the left leg soon followed.

A case of radial paralysis with subsequent paralysis of the arms soon after delivery is reported by Gumpertz.[23]

Flatley[18] considers his case of anterior poliomyelitis in a man 27 years of age, who fell from a wagon and three days later developed a flaccid paralysis of the left upper arm, associated with Bell's paralysis and syringomyelic sensory dissociation, of traumatic origin.

Decroly[14] questions whether the fall caused the paralysis or *vice versa*, in two cases of supposedly traumatic origin.

*Epidemics.*—That the disease is probably of an infectious nature has become widely accepted. Its development and course remind one of an infectious disease. Several distinct epidemics have been reported which have been so widespread that the factor of a mere coincidence can be ruled out without a question.

Caverly[9] recorded a remarkable epidemic which occurred near Rutland (Vermont). During the summer of 1894, 119 cases developed. Eighty-five were under 6 years of age, and 18 died. Macphail[29] described the same epidemic.

A Stockholm physician, Medin[30] has reported an epidemic in which 44 cases occurred in one month in the same town.

In 1888 Rissler[46] had an opportunity of studying the pathological changes occurring in the cord in 5 cases during an epidemic. Leegard[25] narrates a case of an 8-year old boy who developed anterior poliomyelitis; four days later a 20-year old servant came down with the same disease.

Johannessen[24] gives a list of the number of cases occurring in Stockholm from 1888 to 1895. They varied from 3 to 11 in a year, except in 1895, when there were 21 cases—4 in July, 4 in August, 8 in September, 2 in October, 2 in March and 1 in June. In 1889 there were 5 cases in Jena—1 in June and 4 in July; age from 9 months to 3 years.

Pierracini[42] reported an epidemic of 7 children in 15 days in a town in Italy near Florence. Bucelli[6] saw 17 cases within four months.

Auerbach[2] had 15 cases from May to December, 1898; 4 on the same street. He goes as far as to say that the difference between epidemic poliomyelitis, cerebro-spinal meningitis, sporadic or epidemic, and encephalitis, is not one of pathogenesis but merely that of selection of the same micro-organism, the Weichselbaum-Jaeger meningococcus.

Johannessen[24] studied 23 cases from 1893 to 1899, 16 boys and 7 girls. Two cases occurred from March to May; 10 from June to August; 5 from September to November, and 1 from December to February. He thinks that it is possible that some relation exists between poliomyelitis and the gastro-intestinal disturbances to which small children are subject. He quotes Bülow-Hanson and Harbitz, who found swollen mesentary glands and hyperemic Peyer's patches.

In an epidemic of poliomyelitis, Leegard[25] observed 54 cases: 45 of these occurred from July to October; 12 had gastric symptoms. Colds were given as causes in 6 cases; exertion in 7 and both in 9. Heredity seemed to play no rôle. In two districts there was also an icterus epidemic. The epidemic nature of the disease was shown in the fact that it spread along the lines of most extensive travel.

A remarkable family epidemic in which all of 7 children were attacked within 10 days was recorded by Pasteur.[39] Two recovered completely; the others had residuals.

Painter[38] had an opportunity to study an epidemic of 38 cases which occurred in Gloucester (Mass.) within a radius of 4 miles, and most of them near together. This epidemic lasted from June to September.

Colmer[12] reported 8 or 10 cases of teething paralysis, identical with anterior poliomyelitis in West Feliciana (La.).

The following cases are cited by Painter, but no references are given. In 1881 Bergenholtz observed 13 cases in 1½ months. Oxholm reported 9 cases in a small community in Norway in an interval of 6 weeks. Cordier described 13 cases near Lyons (France) in June and July, 1885. In 1896 Brackett reported an epidemic of 8 or 10 cases in North Adams (Mass.). In 1899 5 cases in a population of 49 were observed by Newark. Two fatal cases of cerebro-spinal meningitis occurred during the epidemic reported by Painter. No relation between the cases of anterior poliomyelitis could be established but the symptom-complex in all was very similar. Some cases began during very hot weather; others after a sudden drop of temperature and others after bathing. One case died. The ages of the patients varied from 15 months to 10 years. Eight cases, 2 years or under; 21 cases, 3 years or under; 7 cases, 4 years or older.

Packard[37] reports a case in which two sisters were attacked within 3 days.

J. M. Taylor[56] saw an epidemic in a village of 1,500; 4 cases were seen, 2 being brothers. Two other cases occurred in a neighboring town.

A similar case, in which a 4½-year-old boy developed anterior poliomyelitis one week before his 3-year-old sister, is cited by L. Guinon and Rist.[22]

Lepine[26] observed a case of typhoid myelitis occurring in the course of typhoid fever and probably caused by the bacillus of Eberth. Typical lesions of anterior poliomyelitis were found in the lumbo-sacral cord. The cerebro-spinal fluid was increased in quantity.

According to Gossage[21] the disease attacks strong as well as weak children, and often follows the acute specific diseases of childhood. Convulsions and coma are common initial symptoms in epidemics, but rare in sporadic cases. In Medin's epidemic, two cases of multiple neuritis occurred at the same time with anterior poliomyelitis. The relation of neuritis with anterior poliomyelitis has also been observed by other writers.

An epidemic of neuritis in which two cases of anterior poliomyelitis occurred came under the observation of Eisenlohr.[16]

*Bacteriology.*—The highly suggestive clinical course of the disease, together with the numerous epidemics which have been reported, have prompted some of the more recent investigators to make bacteriological examinations of the cord and cerebro-spinal fluid, while others have attempted to produce the disease experimentally in animals. Both have been partially successful, though a uniformity in results is still lacking. There is as yet no proof that the disease depends upon the action of some specific micro-organism, but sufficient evidence has been accumulated to show that it is dependent upon toxins, of various kinds, either bacterial or other poisons.

Schultze[49] found in one case the Weichselbaum-Jaeger meningococcus by lumbar puncture, and believes the coincidence with meningitis is far from rare.

Dercum[15] made a lumbar puncture in a case of anterior poliomyelitis and found numerous micrococci which morphologically and tinctorially resembled the diplococcus of Sternberg.

Spiller[52] showed the presence of the staphylococcus pyogenes albus in cultures made from the intradural fluid of the cord in a case of paraplegia, resembling transverse myelitis; but it showed at autopsy an extensive poliomyelitis.

Engel[17] made a lumbar puncture in a case of anterior poliomyelitis six days after the onset of the paralysis in a 5-year-old child. No micro-organisms were found in the clear fluid, but cultures showed the presence of the staphylococcus pyogenes albus (patient had had purulent otitis media since the age of 16 weeks).

Chapin[10] described an epidemic in which Dr. Brooks found diplococci in the anterior horns, but not in the blood or meninges. In a second case diplococci were found in the blood during life.

H. Vincent[58] isolated the Eberth bacillus and another one strongly pathogenic for rabbits, from the spleen of a patient who died of typhoid fever. He inoculated several rabbits with a mixture of this culture, and produced a severe enteritis. After this improved, he noticed a rapid paralysis beginning in the hind legs. The animal died 14 days after the onset of the paralytic symptoms. The autopsy showed typical lesions of anterior poliomyelitis in the lumbar cord, absence of cells and thickened vessels. He was, however, unable to produce it at will in other animals. He was unable to isolate the bacilli from the rabbit, and considers the lesion the result of the absorbed toxins from the bacteria and not resulting directly from their presence.

Roger and Bourges[47] produced similar lesions after injecting streptococci. Guinon and Rist[22] made lumbar punctures in the cases referred to above, 14 days after the onset in the boy, and 7 days after the onset in the girl. The cerebro-spinal fluid was sterile and without leukocytes.

Thomas[57] reports two cases of acute ascending paralysis which, however, were considered anatomically as acute anterior poliomyelitis. Bacteriological examination of the sections and cultures were negative. A negative result was also obtained in Bülow-Hansen and Harbitz's case.

Batten[4] made bacteriological examinations of the cerebro-spinal fluid and of the anterior horns of a case of anterior poliomyelitis and found in the cerebro-spinal fluid: (a) a short difficult staining bacillus (probably proteus vulgaris) ; (b) a fat coccus resembling cocci from infected wounds; and (c) many ordinary staphylococci. From the anterior horns he isolated: (a) a short poorly-staining bacillus, but not the same as the other: (b) the same fat coccus but no staphylococci.

In a recent case six weeks after the onset Achard and Grenet[1] found a pronounced lymphocytosis in the cerebro-spinal fluid of a 14-year-old child suffering from anterior poliomyelitis.

In contrast with this observation J. Fraenkel[19] and Niedner and Mamlock[33] found no lymphocytosis in cases of poliomyelitis.

The peculiar course which the disease took in this case reminds one strongly of an acute ascending paralysis. Cases have been reported clinically as Landry's paralysis which at autopsy have shown lesions characteristic of anterior poliomyelitis and should really be classed in this group. Clinically the two conditions are at times inseparable and no hard and fast lines can be drawn between them. With a rapid fatal termination and development of bulbar symptoms, we speak of it as Landry's paralysis. Both the clinical and anatomical findings of the two diseases overlap to such an extent that the too free use of the term "Landry's paralysis" is very confusing. Acute ascending paralysis seems to be more the result of a toxic or infectious process. In many cases splenic enlargement, inflammation of the lymph glands, and albuminuria were noticed. As in poliomyelitis various micro-organisms have been described in the blood and organs.

Bassoe[3] reports a case of acute ascending paralysis in which the lesion was a hemorrhage poliomyelitis. Encapsulated diplococci were isolated from the heart's blood. The bacillus coli communis was found in the cord, spleen and peritoneal fluid. In the liver a pneumococcus and the staphylococcus cereus flavus were found. He also produced experimentally a tigrolysis of the ventral horn cells in a rabbit inoculated with the staphylococcus pyogenes albus. He quotes Mönckeberg, who also found a hemorrhagic poliomyelitis in a typical case of Landry's paralysis.

*Pathology.*—While the literature of poliomyelitis is very extensive and the disease is one pointing to definite and easily recognized changes in the spinal cord, more particularly the gray matter, there are still some points which are by no means clear. The etiological factors are very uncertain and obscure. The reason why the cord alone should be attacked, and just how the infection or intoxication reaches the cord, is in doubt, and it remains a mooted question whether the disease attacks primarily the nervous elements or the vessels.

There are still many who adhere to the teachings of Charcot[11] that the disease is primarily of a parenchymatous nature, in spite of the many strong arguments in favor of a primary vascular change. Charcot made his examinations in cases of long standing after the inflammatory condition had subsided, which explains in part the reason for his conclusions. His was the generally accepted theory until Roger and Damaschino[48] had an opportunity of studying several recent cases from which they concluded that the vascular lesions were probably primary.

Bruining[5] is one of the few recent writers who adheres to Charcot's views. He studied clinically two interesting cases of father and son in whom the disease began in adult life. The father died two years after the onset and the cord showed extensive atrophy of the anterior horn cells. The vessels were congested but there was no perivascular infiltration. Numerous large neuroglia cells, with protoplasm staining red with eosin, were seen in the anterior horns of the dorsal cord. From the absence of any inflammatory changes, the author concludes that the disease attacks the nervous elements primarily.

Bullen[7] reports an adult case in which the pathological appearances were for the most part those of parenchymatous atrophy only.

Nonne[34] likewise considers the cell destruction in the anterior horns as primary. No vascular changes were found in his case, but the examination was made two years after the onset. A few writers (Prætorius[44], Schwalbe[50], Preobrajensky [45], and others) are still uncertain whether the toxin affects vessels or cells primarily, or both. They incline, however, toward a primary vascular involvement. Most of the modern writers take a definite stand in favor of primary interstitial changes and secondary involvement of the nervous elements. Among them may be mentioned Siemerling[51], Dauber[13], Goldscheider[20], Placzek[43], Taylor[55], Mott[32], and Batten[4].

Let us consider for a moment the pathological-anatomical characteristics of the cord in poliomyelitis. The general structure of the gray matter is greatly altered; the vessels are dilated and there is enormous distension of the perivascular spaces with cells of various kinds. There is hyperplasia and hypertrophy of the neuroglia cells and scar tissue is abundant in the subacute and chronic stages. This alteration in structure of the interstitial tissue is sufficient in itself to exert a strong traumatic influence on the nerve cells. Large masses of extravasated cells, and even small hemorrhages, may be seen at times in close contact with the ganglion cells. These cells are as a rule smaller than normal; their prolongations cannot be made out and the chromophilic granules are closely packed together; the cells appear flattened, and cut off as if by pressure. The grade of cell destruction seems to depend on the proximity of the cell to the most extensive interstitial changes. A few cells show a central chromatolysis. They are, however, not as common as the former and in this case only cells in Clarke's column show this tendency. Taylor[55], Siemerling[51], and others speak rather in favor of the mechanical action of the interstitial alterations upon the nerve cells than for toxic influence upon them. It seems not improbable that in the early acute stages the cells may be acted upon by the poisons simultaneously with the vessels; but pathological studies of such cases do not bear this out entirely. The cord shows either a more or less widespread hemorrhagic condition or the presence of a violent inflammation involving more particularly the vessels without any visible structural change of the nerve cells. The widespread scar tissue in the gray matter of some acute cases, such as in the present one, with here and there a fragmented cell, makes one think that the destruction of these cells certainly depends largely on the pressure.

In view of the fact that anterior poliomyelitis may be the result of various poisons, we are hardly justified in attempting to overthrow the theory of a primary parenchymatous involvement entirely, since we know that such poisons as lead, arsenic, etc., are able to produce distinct changes in the cells of the central nervous system. Pathological alterations, typical of anterior poliomyelitis, have been demonstrated in poisoning of various kinds, more especially by lead. These cases might at first lead one to suppose that the nerve cells have a marked affinity for lead and were primarily destroyed by its direct toxic action. However, according to the observations of many, lead causes intense vascular changes which lead to impairment of blood supply and secondary cell destruction. Onuf[36] reports a case of a painter in whom there developed complete flaccid paralysis of both lower extremities, with absent knee jerks. The autopsy revealed probable poliomyelitis of the anterior horns, enormous infiltration of round cells about the vessels, general destruction of the nerve cells and marked involvement of the anterior roots. In experiments with lead on 36 animals, Stieglitz[54] obtained in one guinea pig an acute poliomyelitis with cell infiltration and more or less destruction of nerve cells around the foci. This guinea pig had been paralyzed acutely and death occurred within 24 hours. Vulpian[59] found a pronounced poliomyelitis with colloid degeneration and atrophy of the cells in the case of lead poisoning. Small-celled infiltration and sclerotic islands were present in the roots of the cervical enlargement. He also produced artificial lead intoxication in a dog with paralysis of the extremities and demonstrated an exquisite poliomyelitis. A. Westphal[60] and others agree that the lead acts primarily on the central nervous system, but in its selective action it varies. It may attack the nervous elements, or the vessels, or both. Philippe and Gauthard[41] report a case of anterior poliomyelitis resulting from lead poisoning, and Obrastzoff[35] demonstrated anterior poliomyelitis following arsenical poisoning. Poliomyelitis has occurred during the course of typhoid fever (Lepine), and Leri and Wilson[27] attribute the poliomyelitis in their case to syphilis.

A very important factor in determining the peculiar susceptibility of the cord to various disease processes is its blood supply. The distribution and extent of the inflammatory process seems to depend upon the arterial supply of the gray matter. The gray matter receives its greatest nutrition from the branches of the anterior spinal artery. It divides into two main trunks within the gray matter each of which is again subdivided into an anterior, a median and a posterior branch. In addition, a number of large and constant vessels enter the gray matter from the pia. The most important of these are the vessels entering with the anterior and posterior roots (the anterior and posterior root arteries). Another very constant vessel passes from the pia to the gray matter at the junction of the anterior and lateral columns (antero-lateral artery). Numerous smaller vessels also enter the gray matter from the pia. The anterior and posterior spinal arteries are derived from the vertebrals and are reinforced by branches of the intercostal, lumbar and other arteries. These vessels form transverse anastomoses in the pia mater and pass into the gray and white substance of the cord. Some of the peripheral or pial vessels simply form superficial capillary loops; others are distributed to the white matter. Most of them penetrate into the gray matter and supply the parts not served by the branches of the anterior spinal. A series of small arterioles enter the posterior fissure and give off branches which supply the adjacent parts of the posterior white columns and Clarke's column, where this is found. No one part, however, is supplied by one set of arterioles, nor is any one set of arterioles confined in its distribution to any one white column or group of cells in the gray matter. Within the cord, as within the brain, all the arteries are terminal. They do not anastomose with other arteries, but each one terminates in its own capillary area and supplies no other. The gray matter of the anterior horn is at the extremity of these vessels and the collateral circulation is poor. This factor is certainly of great importance since it offers a favorable field for sluggish circulation and embolism. It has been suggested by some writers that the lumbar and sacral cords are affected so often, because the blood must travel throughout the length of the anterior spinal artery from the vertebrals, apparently disregarding the fact that these parts are reinforced by branches of other vessels.

Various groups of cells at different levels are diseased indiscriminately and do not follow any definite system. If but one group of vessels, the anterior spinal, were diseased, we might consider the variability of the distribution of the lesions to depend upon the irregular distribution of branches of this particular vessel. However, as specimens of this case show, the peripheral vessels are equally if not more constantly involved than the branches of the anterior spinal arteries and areas of anemic necrosis, small hemorrhages and localized destruction of cells, can be seen in the areas served by these vessels, while the parts supplied by the anterior spinal artery are but little involved. This patchy involvement is present in the cervical and lumbar regions only. One very striking phenomenon which the sections of this case show at every level is the fact that the diseased vessel, whether of central origin (anterior spinal) or penetrating through the white matter into the gray matter, shows the perivascular spaces to be infiltrated with cells practically only in the gray matter. If present in the white matter, the infiltration is of slight extent or in the immediate vicinity of the gray matter. Not a single specimen shows any perivascular infiltration about the anterior spinal artery until its branches have reached the gray matter. This helps to emphasize the selective action of the disease primarily on the gray matter of the cord, and the loose physical architecture of the gray matter probably determines this to some extent. In the areas of most pronounced disintegration of the anterior horn the small-celled infiltration is most extensive, in some places leading to small hemorrhages and in others to large masses of lymphocytes closely packed together, but nowhere to actual pus formation.

From the present case it appears that owing to the anatomical peculiarities of the gray matter, it is the gray matter as a whole which is destroyed and with it the nerve cell. The case demonstrates, I believe, that the gray matter of the cord receives rather more nutrition from the peripheral vessels than is generally taught, especially in the antero-lateral regions. The disease, likewise, involves the peripheral vessels to the same extent that it does the anterior spinal artery. Preobrajensky[45] also found the antero-lateral artery diseased.

The views now generally held, that poliomyelitis is primarily due to a diseased condition of the blood vessels, have been materially strengthened by comparatively recent studies, revealing the presence of thrombosis and embolism in the spinal arteries. These observations represent a decided advance in the elucidation of the pathology of poliomyelitis, especially since we know that the collateral circulation within the cord is established with difficulty, and the presence of a thrombus or embolus in one of the smaller branches of the spinal vessels would shut off the blood completely from the part supplied by it. Mott[32] found inflammatory thrombotic stasis of the terminal branches of the anterior spinal artery, ending in flask-like areas of softening. Batten[4] also found a thrombosis of the anterior spinal artery. It has been demonstrated experimentally that occlusion of the spinal arteries produces a chromatolysis of the anterior horn cells, but inflammatory changes of the vessels are absent. Although a thrombosis in anterior poliomyelitis may be primary, the cell changes which are more extensive must depend not only on the cutting off of the blood supply but also upon other factors such as mechanical pressure and toxins.

From the present case, and from a review of the literature on this subject, we may be led to summarize as follows:

1. Anterior poliomyelitis is the result of a primary inflammatory disease of the blood vessels of the cord which may be thrombotic or embolic.

2. The destruction of the ganglion cells is secondary and depends in part upon the deficient blood supply of the diseased area and in part upon pressure and toxins.

3. The pathological changes occurring in poliomyelitis of children and adults are apparently identical and dependent on similar causes.

4. There is sufficient evidence at hand to consider the disease as a rule of an infectious nature, however, not depending upon a specific micro-organism but resulting from bacterial infections of various kinds, and at times from other poisons.

5. The inflammatory changes are present in the peripheral vessels as well as in the branches of the anterior spinal artery, though these changes are seldom visible until the vessels enter the gray matter.

6. The inadequate collateral circulation within the anterior horns is favorable for sluggish circulation and embolism.

## BIBLIOGRAPHY.

[1] Achard, C. H., and Grenet, H. “Poliomyelitis anterior acuta und arachnoidale Lymphocytose.” Abs. in Neur. Centralbl., Vol. 23, 1904, No. 18, p. 875.

[2] Auerbach. Jahrbüch f. Nervenheilk., Vol. 1, 1899, p. 41.

[3] Bassoe, P. “Some Observations on Acute Ascending Paralysis.” Transactions of the Chicago Pathological Society, Vol. 6, Nov. 14, 1904, p. 197.

[4] Batten, F. E. “The Pathology of Infantile Paralysis (acute anterior poliomyelitis.)” Brain, Vol. 107, Autumn, 1904, p. 376.

[5] Bruining, J. “Zwei Fälle von sog. Poliomyelitis anterior acuta bei Vater und Sohn.” Zeitsch. f. Nervenheilk., Vol. 27, 1904, p. 269.

[6] Bucelli. Cited by Johannessen in Festschrift in honor of Abraham Jacobi, May 6. 1900, p. 263.

[7] Bullen, F. St. J. “The Clinical History and Morbid Anatomy of a Case of Anterior Poliomyelitis with Peripheral Nerve Changes.” Jour. of Mental Science, Vol. 38, 1892, p. 71.

[8] Burckhardt, M. “Poliomyelite anterieuse subaigue progressive.” Archiv. de Médicine des Enfants, October, 1902, p. 608. Abs. in Revue Neurologique, 1903, p. 421.

[9] Caverly. New York Medical Record, Vol. 2, 1894.

[10] Chapin. Archives of Pediatrics, November, 1900. Cited by Gossage.

[11] Charcot. Archiv. de Physiol., Vol. 3, 1870, p. 134.

[12] Colmer. Am. Jour. Med. Sciences, 1843. Cited by Painter.

[13] Dauber. “Zur Lehre von der Poliomyelitis anterior.” Deutsch. Zeitschr. f. Nervenheilk., Vol. 4, 1893, p. 200.

[14] Decroly. “Syndrome poliomyélitique succédant à une chute sur le dos.” Jour. de Neur., Vol. 5, 1900, p. 107.

[15] Dercum, F. X. “Note on a Case of Acute Poliomyelitis.” JOUR. NERVOUS AND MENTAL DISEASE, Vol. 27, 1900.

[16] Eisenlohr. Berlin Klin. Wochenschrift, Vol. 24, 1887, p. 781.

[17] Engel, F. “Bakteriologisches Ergebniss einer Lumbalpunktion bei Poliomyelitis anterior.” Prag. Med. Wochenschrift, Vol. 25, 1900, No. 12. Abs. in Centralbl. für Nervenheilk., Vol. 23, 1900, p. 756.

[18] Flatley, J. W. “A Case of Acute Poliomyelitis Occurring in an Adult.” Am. Med., Vol. 8, 1904, p. 956.

[19] Fraenkel, J. “Lymphocytosis of the Cerebro-spinal Fluid.” Med. Record, Vol. 65, 1904, No. 4.

[20] Goldscheider, A. “Ueber Poliomyelitis Anterior.” Deutsch. med. Wochenschrift, 1893, No. 19.

[21] Gossage, A. M. “The Etiology of Infantile Paralysis.” Am. Jour. of Med. Sciences, Vol. 123, 1902, 6. 798.

[22] Guinon, L., and Rist. “Deux cas de poliomyélite antérieuse aiguë sans réaction méningée cytologique chez un frére et une soeur Abs. in Neur. Centralbl., Vol. 23, 1904, p. 769.

[23] Gumpertz. “Ein Fall von puerperaler Poliomyelitis anterior.” Archiv. f. Pysch., Vol. 36, 1903, p. 918.

[24] Johannessen, A. “Poliomyelitis anterior acuta.” Festschrift in honor of Abraham Jacobi, May 6, 1900, p. 263.

[25] Leegard, C. “Förhandlinger og Foredrag paa det 3de norske Lägemöde,” Vol. 188, p. 80. Cited by Johannessen. Norsk. Mag. f. Lägevidensk, 1901, p. 377. Abs. in Neur. Centralbl., Vol. 21, 1902, p. 505.

[26] Lepine. Rev. de Médecine, Nov. 10, 1903. Abs. in JOUR. OF NERVOUS AND MENTAL DISEASE, February, 1904, Vol. 31, p. 138.

[27] Leri, A., and Wilson, S. A. K. “Un cas de Poliomyélite antérieuse aiguë de l’adulte avec lésions médullaires en Foyers.” Nouv. Icon. de la Salpêtrière, November and December, 1904, p. 432.

[28] Von Leyden, E. “Beiträge zur pathologischen Anatomie der atroph-

ischen Lähmung der Kinder und der Erwachsenen." Archiv. f. Pysch. Vol. 6, 1876, p. 271.

[29] Macphail. Brit. Med. Jour., Vol. 2, 1894, p. 1233.

[30] Medin. Verhandl. Med. Cong. Berlin, II., Abt. VI., 37, 1891.

[31] Meyer, E. Poliomyelitis anterior chronica nach Trauma." Münch. Med. Wochenschr., 1901, No. 5. Abs. in Neur. Centralbl., Vol. 20, 1901, p. 268.

[32] Mott, F. W. "Microscopical Examination of the Spinal Cord, Peripheral Nerves and Muscles in a Case of Acute Poliomyelitis. Fatal Termination Sixteen Days after the Onset." Archives of Neurology, 1899, p. 365.

[33] Niedner and Mamlock. "Zur Frage der Cytodiagnose." Zeitschr. f. klin, Med., 1904, 1 and 2. Abs. in Neur. Centralbl., Vol. 23, 1904, p. 862.

[34] Nonne, V. "Poliomyelitis anterior acuta." Deutsche Zeitschr. f. Nervenheilk., Vol. I, 1891, Nos. 1 and 2, p. 137.

[35] Obrastzoff. "Ein Fall von Arsenicalparalyse." Neur. Centralbl., Vol. 21, 1902, p. 278.

[36] Onuf, B. "Report of an Unusual Case of Lead Paralysis with Autopsy." (Meeting of New York Neurological Soc., Jan. 2, 1900.) JOUR. OF NERV. AND MENT. DIS., Vol. 27, 1900, p. 155.

[37] Packard, F. A. "Acute Anterior Poliomyelitis Occurring Simultaneously in a Brother and Sister." JOUR. OF NERV. AND MENT. DIS., April 18, 1899.

[38] Painter, C. F. "Infantile Paralysis; an Epidemic of Thirty-eight Cases." Boston Med. and Surg. Jour., Dec. 11, 1902, p. 633.

[39] Pasteur. Clin. Soc. Trans., Vol. 30, 1897, p. 1438. Cited by Gossage.

[40] Perrin, M. "Poliomyélite antérieuse subaiguë progressive." Archiv. de Méd. des Enfants. October, 1902, p. 608. Abs. in Revue Neurologique, 1903, p. 421.

[41] Philippe and Gauthard. "Beiträge zum Studium des centralen Ursprungs der Bleilähmung." Neur. Centralbl., Vol. 22, 1903, p. 889.

[41] Pierracini. Cited by Johannessen.

[43] Placzek. "Zur pathologischen Anatomie der spinalen Kinderlähmungen." Archiv. f. Psych., 1903, p. 919.

[44] Prætorius, E. "Zur pathologischen Anatomie der Poliomyelitis anterior acuta infantum." Jahr. f. Kinderheilk., Vol. 58.

[45] Preobrajensky, A. "Etude sur la Poliomyélite aigue." Abs. in Rev. Neurologique, 1903, p. 1056.

[46] Rissler, J. Nordiskt Med. Arkiv., XXII., No. 22, 1888.

[47] Roger and Bourges. Cited by Vincent.

[48] Roger and Damaschino. Gaz. Med. de Paris, Vol. 26, 1871, p. 457.

[49] Schultze, F. "Zur Ætiologie der acuten Poliomyelitis." Münch. med.Wochenschr., 1898, p. 1197. Abs. in Rev. Neur., 1899, p. 142.

[50] Schwalbe. "Untersuchung eines Falles von Poliomyelitis acuta infantum im Stadium der Reparation." Beitr. z. rath. Anat., von Zeigler, Vol. 32, 1902, p. 488.

[51] Siemerling, E. "Zur pathologischen Anatomie der spinalen Kinderlähmung." Archiv. f. Psych., Vol. 26, 1894, p. 267.

[52] Spiller, W. G. "A Report of Two Cases of Paraplegia Occurring in Variola, One Being a Case of Anterior Poliomyelitis in an Adult." Brain, Autumn, 1903.

[53] Stark, H. "Ueber Vorderhornekrankungen nach Trauma." Neur. Centralbl., Vol. 23, 1904, No. 14.

[54] Stieglitz, B. (Meeting of New Lork Neurological Soc.) JOURN. OF NERV AND MENT. DIS., Vol. 27, 1900, p. 156.

[55] Taylor, E. W. "Poliomyelitis of the Adult." JOUR. OF NERV. AND MENT. DIS., Vol. 29, 1902, p. 449.

[56] Taylor, J. M. Phila. Med. Jour., Jan. 29, 1898, p. 208.

[57] Thomas, J. J. "A Case of Acute Ascending Paralysis with Autopsy." Am. Jour. of Med. Sc., Vol. 116, 1898.

[58] Vincent, H. "Sur un cas expérimental de poliomyélite infectieuse aigue ayant simulé le syndrome de Landry." Arch. de Méd. Expériment, 1893, No. 3. Abs. in Centralbl. f. Nervenheilk., Vol. 16, 1893, p. 367.

[59] Vulpian. "Maladies du System Nerveux," Paris, 1879. Cited by von Monakow, Archiv. f. Psych., Vol. 10, 1879, p. 495.

[60] Westphal, A. Archiv. f. Psych., Vol. 19, 1887, p. 620.